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Lupine Anthracnose

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Lupine anthracnose was observed in late-planted blue lupine (*Lupinus angustifolius* L.) at Quincy, Fla., in March 1939 (*6*).[2] The causal fungus was found to be *Glomerella cingulata* (Ston.) Spauld. & Schrenck. At that time, lupine[3] was grown largely for experimental purposes and its rapid development into the major winter cover crop in large areas of the Southeast was not foreseen. Because the crop was of little economic importance at the time, work on the disease was not carried beyond determination that under high-humidity conditions *G. cingulata* can attack young leaflets, petioles, stems, and cotyledons of blue lupine seedlings and the leaves of white lupine (*L. albus* L.). A few years later, as lupine production expanded, work on the anthracnose was resumed at the Georgia Experiment Station, Experiment, Ga. The results of the later investigations are reported in this paper.

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[1] The investigations reported here were made cooperatively by the Division of Forage Crops and Diseases, Bureau of Plant Industry, Soils, and Agricultural Engineering, Agricultural Research Administration, U. S. Department of Agriculture, and the Georgia Experiment Station, Experiment, Ga. (Georgia Experiment Station Journal Series Paper No. 211).

[2] Italic numbers in parentheses refer to Literature Cited, p. 17.

[3] Unless otherwise stated, the name "lupine" in this paper refers to blue lupine (*Lupinus angustifolius* L.).

DISTRIBUTION AND IMPORTANCE

Specimens of anthracnose-infected lupine have been collected in Florida, Georgia, Alabama, North Carolina, and Texas. Probably the causal fungus occurs in most lupine-growing sections of the southeastern United States. In general the disease is serious only in parts of southern Georgia, southern Alabama, and northern Florida. Even within these areas it varies greatly in importance from year to year. Usually it is not serious except in wet seasons. Its potential importance was suggested by Wilson (*9*), who stated in 1949 that the lupine acreage in Baldwin County, Ala., had decreased sharply because of anthracnose and predicted that the crop would not be planted in that locality to any great extent in the future.

SYMPTOMOLOGY

When the author's previous paper on lupine anthracnose (*6*) was written, little was known regarding the symptoms of the disease other than those on seedlings. Although Decker (*2*) and Wilson (*8*) have since given brief descriptions of the disease on older plants, a more detailed discussion of this subject seems to be desirable.

The lesions on the cotyledons of young seedlings appear as very small pits, about 1 mm. in diameter and more or less circular in outline (fig. 1, *A*). These lesions enlarge rather rapidly, soon becoming one-third to one-half the width of the cotyledons. Some are uniformly a buffy brown;[4] others have a citrine-drab background and a light-brownish-olive center. As the cotyledons wither, a definite raised buffy-brown border appears around most of the lesions. Sporulation may or may not occur on the lesions, depending on temperature and moisture conditions.

Lesions on young stems (fig. 1, *B*) are ochraceous-tawny to cinnamon-brown but darken with age to nearly black. These lesions either are sunken from the first or become so as the dead tissue dries and shrinks. Both conidia and ascospores may be present on lesions in the field when conditions are favorable for development of the fungus. The number of acervuli or of perithecia present is highly variable. Setae often are abundant but may be entirely lacking. On young stems the lesions vary from 1 mm. to 10 cm. in length. On older stems they may be longer (fig. 1, *C*) and often extend nearly or entirely around the stem. A large proportion of the total surface area of the stem and branches may become involved. Maturing plants so extensively affected that much of the stem and branch tissue is discolored may nevertheless survive with little or no apparent damage. There may be concentric rings on the surface of the canker, and it may be covered with fruiting bodies. Lesions on older stems are usually lighter in color than those on younger ones, sometimes being only slightly darker than the adjacent healthy tissue.

Under usual field conditions lupine leaves are not seriously affected. Leaf lesions may appear as minute blackish dots less than 0.5 mm. in diameter or as dead brownish areas several centimeters in diameter. Typical leaf lesions are slightly sunken. They are usually circular but are more or less irregular in shape when marginal (fig. 1, *D*).

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[4] All specific color names are those of Ridgway (*4*).

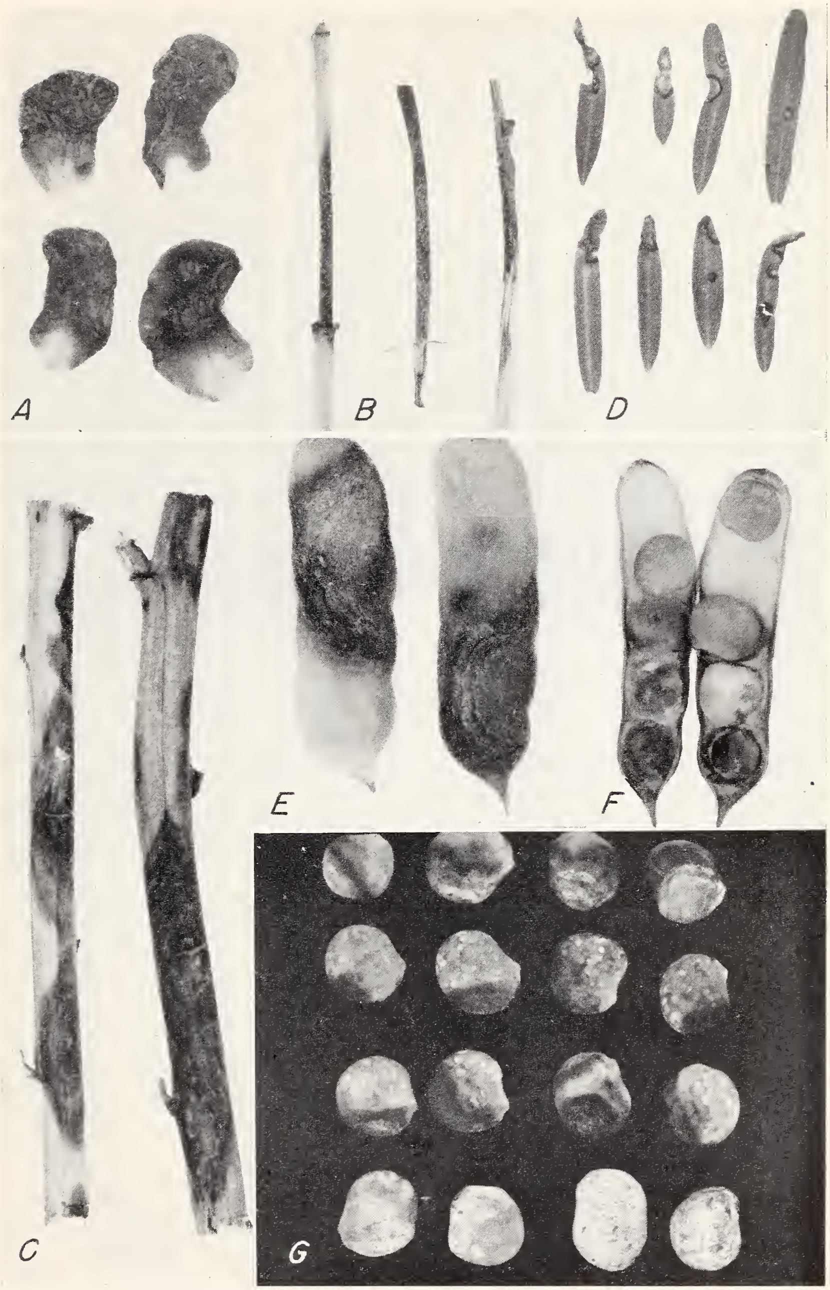

FIGURE 1.—Symptoms of anthracnose on blue lupine. (Except as indicated, views are natural size.) *A*, Circular lesions on cotyledons from inoculated seedlings. × 2. *B*, Blackish cankers on young stems. *C*, Cankers on stems of nearly mature plants. *D*, Lesions on leaflets. *E*, Lesions on pods. *F*, Infected pod opened to show decayed seeds beneath lesions such as those in *E*. *G*, Lesions of different types on seeds. Seeds in the lowest row are badly decayed and are coated with white mycelium. × ⅗.

Most leaf spots are uniformly almost black, but some have centers of a light color approaching buffy brown bordered with the darker color Chaetura drab. Under conditions of high humidity, fruiting bodies of the causal fungus may be present. Diseased leaflets usually fall from the plant prematurely, and considerable defoliation may result. Petioles may become infected and may be girdled in about 96 hours thereafter. Lesions on the petioles resemble those on the young stems.

Young pods are very susceptible and may become heavily infected. At first the pod lesions are very small brownish pits. These enlarge with the result that often, merging, they involve half or more of the pod (fig. 1, *E* and *F*). Usually there are concentric rings on the surface and the fungus fruiting bodies are more or less abundant. As the plants mature and lose their leaves, the diseased pods with their large dark-brown to nearly black lesions become conspicuous. Seeds beneath pod lesions often are decayed and covered with a white mycelial mat (fig. 1, *F*). Most of the seeds thus affected are badly decayed; many of them consist of nothing more than remnants of decayed tissue, interspersed with mycelium, and the seed coat. Seeds less seriously affected can be identified by the presence of a discolored area on the seed coat. The discolored area may be barely large enough to be seen with the unaided eye; on the other hand, the whole seed coat may be discolored (fig. 1, *G*). The lesions vary in color from dusky neutral gray to blackish slate to nearly black, sometimes shading off to Sudan brown or Brussels brown at the edges. This description does not distinguish seeds affected with anthracnose from those similarly affected by the brown spot fungus (*Ceratophorum setosum* Kirchn.).

Little information is available regarding symptoms of anthracnose on yellow lupine (*Lupinus luteus* L.) and white lupine (*L. albus* L.) under field conditions in this country. Yellow lupine, which is grown to some extent in Florida, southern Georgia, and southern Alabama, has not been observed under conditions favorable for anthracnose infection. Lesions on young yellow lupine plants inoculated under greenhouse conditions occurred principally on the leaves and petioles. The lesions were closely similar to those observed on corresponding parts of blue lupine under similar conditions. Often the plant was entirely killed as a consequence of the passage of the fungus from the petiole into the stem or was greatly stunted because of the loss of its leaves. White lupine sometimes is seriously injured or killed. The infected plants have large blackish stem lesions and many large dark-brown leaf lesions, and the petioles may be girdled.

THE CAUSAL FUNGUS

MORPHOLOGY

The conidia of the fungus *Glomerella cingulata* are unicellular, hyaline, and straight or tapering slightly at both ends, and measure $3.5\text{–}6.3\mu \times 8.4 - 18.9\mu$ (average, $4.6\mu \times 13.3\mu$). Setae, which may or may not be present, are tapering and nearly hyaline above, becoming darker below. They are usually $30\text{–}75\mu$ long and 1–3 septate. Ascospores, which have not been described previously on lupine, are unicellular, allantoid, and usually slightly curved, and are hyaline when young to slightly lemon-colored when old. Fifty ascospores from lupine growing in the greenhouse measured $4.6–6.3\mu \times 12.6–18.9\mu$ (average, $5.7\mu \times 15.5\mu$), and 50 ascospores from lupine plants [5] collected in North Carolina measured $4.2–5.3\mu \times 12.6–18.9\mu$ (average, $4.8\mu \times 16\mu$). The measurements of the conidia and those of the ascospores are well within the ranges given for these spore forms of *G. cingulata* by Shear and Wood (*5*), and no character noted gives any basis for distinguishing the fungus on lupine as another species.

PATHOGENICITY

The pathogenicity of *Glomerella cingulata* on blue and white lupine has been discussed by the author in an earlier paper (*6*). Frequent inoculation experiments have demonstrated that some strains of yellow lupine are highly susceptible, but others do not seem to be readily infected. Under conditions favorable for development of the fungus (temperatures of 20° to 30° C. and high moisture), blue lupine can easily be infected by spraying the plants with a suspension of spores or macerated mycelium and holding them in a moist chamber for 48 hours, by inoculating the seed with spores before planting, by injecting inoculum into the cotyledons or stems with a hypodermic needle, by immersing the seedlings in a spore suspension—or, in fact, by any method of inoculation in which the fungus is brought into contact with the host. It has already been reported that the fungus from lupine will rot fruits of apple (*Pyrus malus* L.) (*6*) and peach (*Prunus persica* (L.) Batsch) (*7*). Efforts were made in vain to infect vines of watermelon (*Citrullus vulgaris* Schrad.) and cantaloup (*Cucumis melo* L.). Likewise, infection was not obtained in Chamaecrista (*Chamaecrista procumbens* (L.) Greene), pea (*Pisum sativum* L.), or peanut (*Arachis hypogaea* L.). Coffeeweed (*Cassia tora* L.) and crotalaria (*Crotalaria spectabilis* Roth.) were infected. In these two species small, very dark brown lesions up to 1 mm. in diameter developed, largely on the young cotyledons of seedlings, and the fungus was recovered from these lesions. Infected coffeeweed plants have been found near diseased lupine plants growing in the field.

No very detailed study of pathogenic strains of *Glomerella cingulata* has been made. Infection of lupine was obtained with isolates of the fungus from lupine collected in Georgia, Florida, Alabama, Texas, and North Carolina without bringing out any evidence of striking difference in pathogenicity. Distinct differences were evident, however, when lupine plants were inoculated under greenhouse conditions with isolates of *G. cingulata* from six other hosts; on the lupine an isolate from horsechestnut (*Aesculus hippocastanum* L.)[6] was moderately pathogenic, isolates from sorghum (*Sorghum vulgare* Pers.)[6] and *Arachis* sp. were vigorous parasites, isolates from black medic (*Medicago lupulina* L.) and kudzu (*Pueraria lobata* (Willd.) Ohwi) were weakly pathogenic, and an isolate from cowpea (*Vigna sinensis* (Torner) Hassk.) was nonpathogenic.

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[5] Sent to the writer by S. G. Lehman.

[6] Culture supplied by E. S. Luttrell.

Possible Sources of Inoculum

In the southeastern United States the lupine seed crop is harvested in May or June and is dried and stored until planting time in warehouses or in outbuildings on farms. Although the largest acreage is planted in October and November, planting dates extend from late September to late December or, rarely, into January. This means that the anthracnose fungus, to perpetuate itself on lupine, must survive on or in the seed or in the field through 4 to 7 months including several hot months. Experiments were made to determine in what way inoculum of this fungus reaches new crops of lupine and how it can be kept from reaching them.

SEED

As has been pointed out under "Symptomology," seeds often become infected and many decay badly before the pods mature. Because the badly decayed seeds are shrunken and light in weight, most of them are blown or screened out during the cleaning process. Seeds less seriously damaged are not removed in the cleaning process, and these can carry the fungus to the field (*1*).

Seed lots of the 1947, 1948, and 1949 crops known to be infected with anthracnose were obtained from commercial concerns after they had been stored or were collected from farmers at harvesttime and held in storage for various periods, and repeated tests were made on these lots to determine how long the fungus remained alive in the seed. In general, the seed had been in commercial storage houses from harvesttime until they were brought to the laboratory. Thereafter, most of them were held at laboratory temperature, which is very favorable for seed storage, until studied. In some cases a quantity of seed was held in a refrigerator most of the time after harvest. Tests were made after 3 weeks to 2 years of storage. All the seeds used in these tests had anthracnose lesions. The results are presented in table 1.

On May 26, 1948, a sample was taken from a lot of seed harvested in the spring of 1947 and isolations were made from this sample. Approximately a year after harvest, the fungus was still alive in 8 percent of the seeds. By January 1949 the fungus was dead in all the 3 lots of 1947 seed then studied. It was still alive in large percentages of 2 lots of 1948 seed tested in August 1948. In 1 of 7 lots of 1948 seed tested in January 1949 the fungus was alive in 1 seed out of 48, and in another it was alive in 4 seeds out of 484. The fungus was not recovered from any of the 3 lots of 1948 seed tested in September and November 1949. Nearly all the seeds of lots harvested in May 1949 and 1950 contained the living fungus 3 or 4 weeks after harvest. Viability of the fungus in 1949 seed was noticeably lower in July 1949, and by November of that year the fungus was dead in all seeds of a 1949 lot that had been stored in a commercial storage house at Vienna, Ga. In the 2 other 1949 lots tested at the same time the fungus was still alive in 28 percent and 22 percent of the seeds, respectively. On June 20, 1950, the fungus was dead in all the remaining 1949 seed held at laboratory temperature; but on March 21, 1951, it was alive in 100 percent of the 1949 seed still held in the refrigerator.

Table 1.—*Longevity of Glomerella cingulata in blue lupine seed*

Seed crop [1] and date of test	Seeds used	Seeds yielding fungus
1947 seed:	*Number*	*Percent*
May 26, 1948	25	8
Jan. 14, 1949	50	0
	31	0
	32	0
1948 seed:		
June 16, 1948	50	40
Aug. 4, 1948	18	72
Aug. 10, 1948	17	35
Nov. 29, 1948	25	0
	25	0
	25	0
Jan. 14, 1949	45	0
	50	0
	46	0
	48	2
	50	0
	48	0
	484	.8
Sept. 14, 1949	100	0
	30	0
Nov. 4, 1949	50	0
1949 seed:		
June 17, 1949	190	100
	190	100
July 28, 1949	200	79
Aug. 3, 1949	200	75
Aug. 15, 1949	50	56
Sept. 14, 1949	[2] 100	99
	100	31
	100	44
Sept. 30, 1949	[2] 20	100
Nov. 5, 1949	50	28
	50	22
	50	0
June 20, 1950	50	0
	50	0
	50	0
	50	0
	[2] 50	86
Mar. 21, 1951	[2] 200	100
1950 seed: June 20, 1950	50	98

[1] Lupine seed is harvested in May or early June.

[2] This lot had been held in a refrigerator most of the time since harvest.

These data show that the fungus in the seed gradually loses its viability in commercial storage. By planting time of the harvest year the fungus in approximately half the infected seed held under ordinary commercial warehouse conditions had died. Although the samples tested have been limited in number and size, the consistent trend shown by the results indicates that seed held under the usual storage conditions until the second planting period after their harvest will then be largely if not completely free of the anthracnose fungus.

On June 14, 1948, a sample of 100 seeds was taken from each of 6 lots of infected seed harvested in 1947. Each sample was placed in a 125-ml. flask, 10 ml. of distilled water was poured over it, and the flask was shaken frequently over a period of 30 minutes. About 0.5 ml. of the water was then pipetted off, placed on a slide, and studied under the microscope, and 1 ml. was plated on corn-meal agar. In no case were any spores of *Glomerella cingulata* found. Several other experiments of this nature were run. Few spores of *G. cingulata* were found on the seeds, and all these proved to be dead. These results indicate that the anthracnose fungus probably is not commonly carried to the field in the form of spores on lupine seed surfaces if planting is done with seed of a previous year.

OLD PLANTS

Lupine seed is harvested with a combine, and the old stems are left on the ground. Because the seed crop matures rather late, the land sometimes is not worked until autumn. Attempts to isolate the fungus from cankers found on the stems in one unworked field in October failed. Portions of the stems were cut into small segments and placed on soil in pots in which lupine seed had been planted. No infection resulted. Early in July 1948, up to 25 percent of the conidia taken from cankers on different parts of diseased plants growing in the open germinated. When the test was repeated on August 3, 1948, only a few conidia germinated but mycelium grew from some of the heavy-walled basal cells of the acervuli. These results strongly suggest that the conidia on old stems left in the field do not live through the summer but that some of the heavy-walled cells making up the hymenial layer may survive longer than the spores.

Although no positive evidence was obtained that the fungus can survive to planting time in stem cankers under field conditions, a possibility that it does so under certain favorable conditions is suggested by the fact that it survives in dry seeds for several months.

SOIL

In June 1948 a 50- by 6-foot hotbed was planted with seed that had been inoculated by dipping them in a heavy spore suspension. Before the end of the summer all the plants had died of anthracnose. On October 26, 1948, the hotbed was replanted with seed of the lupine grown commercially in the Southeast and a number of imported lots. Although the anthracnose fungus had been so abundant in the hotbed a few weeks before that no plant survived, no anthracnose appeared on any of the plants of the new crop. Likewise, none appeared on any plant of a crop grown in the hotbed the following spring. Although not conclusive, these results support the theory that the fungus does not live through the summer on dead plant parts or in the soil in the absence of the living host. In the autumn of 1949, 5 pounds of lupine seed known to be free of the anthracnose fungus was planted on soil on which lupine had never been grown, to determine whether the fungus was present in the soil. Likewise a lot of commercial lupine seed a considerable proportion of which was known to be infected with *Glomerella cingulata* was planted in a field where lupine had been grown the preceding year and many of the plants had been affected with anthracnose. Rainfall was low during the autumn and winter. No anthracnose appeared in either planting. This result shows that under weather conditions unfavorable to the anthracnose fungus it is not impossible to obtain a healthy crop of lupine from diseased seed planted on soil that produced a badly diseased crop of lupine the previous season.

PHYSIOLOGY

THERMAL DEATH POINT

The effect of heat on the fungus used in these experiments was determined by drawing conidia from pure culture, suspended in sterile tap water, into sterile fine capillary tubes and sealing the tube openings with 60° C. paraffin, fastening each of the tubes to the bulb of a thermometer with a rubber band, and immersing the thermometer and tubes for different lengths of time in water held at various high temperatures. After treatment the tubes were held in cold water for a few seconds, then their contents were poured onto agar in petri dishes. Three tubes of suspension were used for each test, and each test was replicated three times. With few exceptions the fungus was killed in 10 minutes at 50°, and it was always killed in that time at 51° or above. The data for the 10-minute tests are as follows:

Temperature (°C.):	*Growth of fungus after treatment*
48	+
49	+
50	+
51	0
52	0
Unheated control	+

At 50°, only a few spores germinated in one out of nine tubes.

LONGEVITY OF CONIDIA

The viability of conidia taken from pods collected in the field on May 5 and May 25, 1948, was tested by placing them in a culture solution in hanging drops and on water agar in petri dishes. Conidia from a pure culture were used as a control. Of the conidia in the control, 98 percent germinated. Of those taken from pods collected on May 5 and tested on June 16, 1948, none produced normal germ tubes. A very few of those from the pods collected May 25 germinated, forming short, stunted, abnormal germ tubes; most of them were dead. These results showed that the thin-walled conidia formed on the pods before harvest were largely dead about a month after harvest, and indicate that such spores probably play little if any part in carrying the fungus through the summer and until planting time. (The possibility that new conidia or ascospores may be formed in old lesions on plant debris lying in the field has not been exhaustively investigated.)

To test the longevity of the conidia further, drops of suspensions of conidia from the host and from pure culture were placed in water on sterile cover glasses held in sterile petri dishes and the water was allowed to evaporate. At regular intervals cooled melted agar was poured in one or more of the plates and the cultures were incubated and studied. Under the dry conditions of this experiment, all the conidia were dead in less than a month.

TEMPERATURE RELATIONS IN CULTURE

To determine the approximate maximum, optimum, and minimum temperatures for growth of the fungus, pieces of potato dextrose agar about 5 mm. square on which the fungus was growing in petri dishes were placed in the centers of other petri dishes containing potato dextrose agar. Four dishes were inoculated with each of two isolates of the fungus from lupine and held at different temperatures for a period of 5 days. The longest, shortest, and average diameters of the colonies developed at each temperature were then determined, and the average diameters were used to calculate total areas. With the average area for the colonies developed at 24.9° C. taken as 100 percent, all the averages were converted to percentages, which are as follows: 4.2°, 0; 14°, 8.1 percent; 19.7°, 57.5 percent; 24.9°, 100 percent; 29.8°, 65.7 percent; 34.9°, 0.1 percent; 39.5°, 0. At the end of 5 days the cultures that had been held at 39.5° were exposed to a temperature of 25° for 48 hours. The fungus made no growth. This showed that it was dead. In a similar experiment the fungus made very slight growth but was not killed in 5 days at 37.5°. The cultures held at 4.2° made no growth in 13 days, but resumed growth at 25°.

These results show that for the strain of *Glomerella cingulata* used the temperature most favorable to growth is near 25° C., survival over a 13-day period is possible at 4.2°, and the maximum for survival over a 5-day period is between 37.5° and 39.5°. This maximum agrees fairly well with that found by Edgerton (*3*) for his strain of *G. cingulata;* but the optimum for his strain was 3° higher than that indicated for the isolates from lupine.

CONTROL

Although lupine anthracnose is very serious in limited areas during some years, it is not destructive enough at this time over the lupine belt as a whole to justify widespread control measures.

Evidence has been presented above that lupine seed can be freed of the fungus by holding it in storage until the second planting time, or for about 18 months. This measure entails considerable added expense, however, and probably will not be acceptable to growers unless in emergencies. With this in mind, an effort was made to find a more practical seed treatment.

SEED TREATMENT

HOT WATER

Experiments were conducted to determine whether the anthracnose fungus within lupine seed could be killed by immersing the seed in hot water. Seeds noticeably infected with the anthracnose fungus were tied in cheesecloth bags in lots of 25 each. They were given a 1-hour presoaking at 45° C., because preliminary experiments had shown that this was helpful in getting a quick kill of the fungus and did not injure the seed. The bags of seed were then immersed in large pans of water each of which was held at a constant temperature, and were agitated constantly during a certain immersion period. At the end of that time the seed were placed in cold water for a minute or longer. Then they were disinfected for 10 seconds with 95-percent alcohol, dipped in a 1–1,000 solution of $HgCl_2$ for 1 minute, washed in sterile water, and plated on potato dextrose agar. In each test 50 healthy seed were treated in the same manner as the diseased seed except that they were germinated instead of being plated on agar. Two controls were run, one with untreated seed and the other with seed given the presoaking treatment only.

Data showing the reactions of healthy and diseased seed to immersion in water at 50°, 51°, and 52° C. for various periods are given in table 2. The healthy seed to which only the presoaking treatment was given germinated 100 percent; those not treated, 96 percent. Exposure of healthy seed to a temperature of 50° for 30 minutes, the time necessary for this temperature to kill the fungus in the seed, reduced germination to 20 percent. Healthy seed that had been held in water at 51° for 20 minutes germinated only 48 percent, and this exposure was not sufficient to eliminate the fungus from the diseased seed. Immersion in water at 52° for 10 minutes killed the fungus in all the diseased seed but reduced the germination of the healthy ones by approximately half. Hot-water treatments effective against the fungus cause too great losses in germination to be practical.

Table 2.—*Effect on germination and on infection with Glomerella cingulata of presoaking lupine seed for 1 hour in water at 45° C. and then soaking them at higher temperatures*

Treatment		Germination of healthy seed	Diseased seed in which the fungus remained alive
Presoaking plus—		*Percent*	*Percent*
Soaking at 50° for	5 minutes	98	68
	10 "	76	56
	15 "	68	36
	20 "	46	16
	25 "	36	4
	30 "	20	0
	45 "	14	0
Soaking at 51° for	5 minutes	96	35
	10 "	80	23
	15 "	68	21
	20 "	48	4
Soaking at 52° for	5 minutes	98	12
	10 "	48	0
	15 "	28	0
	20 "	8	0
Presoaking treatment only		100	91
No treatment		96	100

HOT AIR

In another series of experiments, seed containing the anthracnose fungus were placed in screen-wire containers, each of which held 50 seeds in a single layer, and were exposed to hot air in a blower-type oven. The containers were suspended in the center of the oven in such a manner that the hot air blew around each seed as evenly as possible. A thermometer and thermocouples were placed in the air stream with the seed, and frequent checks throughout the experiment showed that the temperatures remained practically constant. Healthy seed treated in the same manner as the diseased served as controls. These and a nonheated sample of healthy seed were germinated. The heated diseased seed and a nonheated control were disinfected by the method described above and plated on potato dextrose agar. The results of these experiments are given in table 3. (Numerous preliminary experiments, the results of which are not given in the table, had been made to determine approximately the critical temperature and heating time.)

TABLE 3.—*Effect on germination and on infection with Glomerella cingulata of exposing lupine seed to air temperatures of 70° and 75° C.*

Heating time (hours)	Germination of healthy seed [1] after treatment at—		Seed remaining infected after treatment at—	
	70°	75°	70°	75°
	Percent	*Percent*	*Percent*	*Percent*
0	100	100 100	100	100 98
1		94 100		50 8
2		100	96	6 2
3		100	62	0
4	98	90	46	0
5			40	0
6			20	0
7	98	90	22	0

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[1] All healthy seed, before being tested, were dried to 11-percent moisture. All hard seed were considered to be viable and were counted with those that had germinated.

An air temperature of 70° C. did not kill the fungus within all seed even in 7 hours. A temperature of 75°, in two tests, killed the fungus within 94 and 98 percent of the seed in 2 hours; it eliminated the fungus in 3 hours. Healthy seed exposed to a temperature of 75° for 2 or 3 hours germinated 100 percent. Exposure to this temperature for 7 hours reduced their germination only by 10 percent. One precaution proved necessary; the seed had to be dried to approximately 11- or 12-percent moisture content before they could withstand such heat.

In commercial driers, a temperature of from 40° to 45° C. is commonly used for drying lupine seed; one of 49° is sometimes used if the seed is close to the desired dryness at the start. Since heat so much greater than that commonly used is required to kill the anthracnose fungus, and since the seed may be injured by such heat if they have not been dried with great care, the data presented here can be considered suggestive only. It might be practical to raise the temperature in the drier gradually to 75° for the last 2 or 3 hours of the drying period, thus not only eliminating the anthracnose fungus but also shortening the total drying time required. Much more testing must be done, under conditions more nearly approaching those in the commercial drier, before this method of control can be recommended.

Certain complications arise when lupine seed are heated. Heating dry seed at 75° C. for 2 hours slightly retards germination, and longer heating causes greater retardation. Likewise, as much as 10 percent of the seed may become hard after prolonged heating. Hard seed do not take up water readily, and their germination may be delayed for a few hours or much longer. Hardness of seed brought on by heating gradually disappears in storage.

The fact that the germination of seed heated 7 hours equaled that of seed heated 4 hours indicates that little danger of serious injury would be involved if seed being treated with hot air for control of anthracnose were to remain at 75° C. for a period considerably longer than that necessary to kill the fungus. More of the seed would, however, become temporarily hard.

Rotation

No experiments have been conducted to determine the effectiveness of crop rotation in controlling lupine anthracnose. The results of experiments presented in this paper indicate that the fungus can live for several months in the seed, but suggest that the conidia and the mycelium in mature stems left on the field do not live through the summer and that the fungus does not live long in the soil. These experiments, however, represent too limited a set of conditions to permit drawing any general conclusions. Observations on other lupine diseases indicate that rotation reduces their severity but does not eliminate them. It seems probable that the same thing is true for anthracnose. The fact that *Glomerella cingulata* attacks so many hosts (*5*), including peaches and even some weeds, may limit the effectiveness of rotation, since the inoculum may increase on other hosts during the period when lupines are not grown.

Other workers have expressed the opinion orally that anthracnose of lupine originates from fungus in the soil more commonly than from fungus in the seed. The writer's present information neither confirms nor refutes this opinion. Regardless of the probability that rotation will not be completely effective in controlling the anthracnose, it seems worth while to recommend a 3- or 4-year rotation, which may give partial control of anthracnose and of other diseases.

Spraying or Dusting

The hope of success in controlling anthracnose of lupine under field conditions by spraying or dusting has seemed so remote that no spraying or dusting has been done. The fungus may kill many plants in the autumn and may attack older stems, pods, and seeds in the spring. An entirely satisfactory spray program, therefore, would have to provide protection for the seedlings in the autumn until the activity of the fungus is checked by low temperature. It is possible that the fungus remains active throughout some warm winters, especially in the southern part of the lupine belt. Probably it would be necessary to keep the plants covered for 2 months or longer in the spring to provide adequate protection for the pods and seeds alone. The fact that this is a relatively long period and one of rather high average rainfall tends to discourage spraying and dusting. It seems logical at present to plow under for green manure any field of lupine in which anthracnose has become widespread, rather than to save it for seed.

RESISTANT STRAINS

A number of experiments were conducted in which blue lupine seed were inoculated with a heavy spore suspension before they were planted, or seedlings were inoculated, in an effort to learn whether there is any variation in anthracnose resistance within the lupine strain now grown in the Southeast. In most tests very few of the plants approached maturity and all eventually died of anthracnose. The results of the tests indicate that there is little chance of finding a satisfactory degree of resistance within this strain.

Next a search was made for resistance in 45 introductions and commercial varieties of principal lupine species, chiefly of *Lupinus angustifolius*, *L. luteus*, and *L. albus* (table 4). In 3 experiments 1 or more tests were made on each of these and on the blue lupine grown commercially in the Southeast and 5 selections from it. According to the quantity of seed available, 10 to 50 seeds of each lot were planted. Several methods of inoculation were used, including spraying the plants with a spore suspension or a suspension of blended mycelium, inoculating the seed before planting by dipping them into a heavy spore suspension, and injecting spore suspension into cotyledons or stems with a hypodermic needle.

The incidence of infection varied with the moisture conditions, even when the inoculum was injected directly into the tissue. Usually 100-percent infection was obtained if inoculated seed of susceptible plants were planted in moist soil and there was sufficient moisture to permit infection, sporulation, and spread of the fungus.

All plants of the commercial blue lupine and the 5 selections from it proved extremely susceptible. Under conditions which brought about rapid death of these plants, only minute lesions developed on the stems, cotyledons, and leaflets of plants of several imported lots of blue lupine. Lesions large enough to girdle and kill the plant developed on an occasional plant in some of these imported lots; but usually no more than a few lower leaflets were shed and any infection disappeared with the return of dry weather.

Six of the most resistant lots of blue lupine and one of yellow were retested. Because the hypodermic-needle method of inoculation had given the most uniform infection, this method was used in retesting. Of the plants of the commercial blue lupine used as susceptible controls, 100 percent developed large cankers and most were girdled. For the most part, plants of the imported lots developed no lesions at the point of inoculation. The stems of a few plants of some of the imported lots developed small cankers, but very few were girdled.

The results of these tests (table 4) indicate that under existing conditions the following imported lots, although not immune, are highly resistant (rating of 1 or less) to anthracnose in the early stage of growth: Blue lupine P. I. 167938, P. I. 167939, P. I. 167941, P. I. 167942, P. I. 167943, P. I. 168526, P. I. 168528, P. I. 168529, P. I. 168532, P. I. 168533, P. I. 168534, P. I. 168535, P. I. 168536, and P. I. 180709; yellow lupine P. I. 168539 and P. I. 168540; and *Lupinus hispanicus* P. I. 168537.

Table 4.—*Anthracnose resistance of strains and P. I. lots of 6 lupine species, rated on a 0–5 scale,[1] in 1 to 4 tests made in the early stage of growth*

Species and strain or P. I. lot	Plants used	Resistance ratings	Average resistance rating
Lupinus angustifolius:	*Number*		
Commercial blue and selections	534	4, 5, 5, 5	4. 8
P. I. 167938	85	–, 1, 1, 1	1. 0
P. I. 167939	51	1, 1, 1, –	1. 0
P. I. 167940	105	1, 1, 1, 2. 5	1. 4
P. I. 167941	35	1, 1, 1, –	1. 0
P. I. 167942	38	0, 1, 1, –	. 7
P. I. 167943	104	0, 0, 1, 1	. 5
P. I. 167944	7	5, 5, –, –	5. 0
P. I. 168526	62	1, 1, 1, –	1. 0
P. I. 168527	44	1, 2, 1, –	1. 3
P. I. 168528	60	–, 0, 1, –	. 5
P. I. 168529	56	0, 1, 1, 1	. 8
P. I. 168530	38	0, 1, 2, –	1. 0
P. I. 168531	56	0, 3, 1, –	1. 3
P. I. 168532	48	–, 1, 1, –	1. 0
P. I. 168533	34	1, 0, 1, –	. 7
P. I. 168534	96	–, 0, 2, 1	1. 0
P. I. 168535	94	–, 1, 1, 1	1. 0
P. I. 168536	68	0, 1, 2, –	1. 0
P. I. 168892	27	4, 5, –, –	4. 5
P. I. 180706	7	–, 5, –, –	5. 0
P. I. 180707	15	–, 5, –, –	5. 0
P. I. 180708	22	–, 3, 1, –	2. 0
P. I. 180709	42	–, 1, 1, –	1. 0
Sweet blue	19	–, 5, –, –	5. 0
L. luteus:			
P. I. 168538	1	0, –, –, –	0
P. I. 168539	104	2, 1, 1, 0	1. 0
P. I. 168540	4	0, 0, 1, –	. 3
P. I. 168541	32	2, 4, –, –	3. 0
P. I. 168542	32	2, 4, –, –	3. 0
P. I. 168544	16	1, 3, –, –	2. 0
P. I. 168545	11	3, –, 1, –	2. 0
P. I. 180711	16	–, 5, –, –	5. 0
P. I. 181993	19	–, 5, –, –	5. 0
White-seeded yellow	38	–, 5, –, –	5. 0
L. albus:			
P. I. 168891	30	3, 3, –, –	3. 0
P. I. 170524	10	–, 3, –, –	3. 0
P. I. 170528	21	3, 5, –, –	4. 0
P. I. 177455	30	3, 3, –, –	3. 0
P. I. 177456	32	4, 2, 2, –	2. 7
P. I. 179361	32	5, 5, –, –	5. 0
P. I. 181775	26	4, 5, –, –	4. 5
Hastings White	21	–, 4, –, –	4. 0
L. hispanicus: P. I. 168537	12	0, 1, –, –	. 5
L. hirsutus: P. I. 170525	1	1, –, –, –	1. 0
Lupinus sp.: P. I. 179523	27	4, 5, –, –	4. 5

[1] The highest rating is 0. A dash signifies omission of the lot from a test.

Limited tests made at a later stage of growth indicate that the pods and seeds of three of the lots just listed, P. I. 167938, P. I. 168529, and P. I. 168535, do not possess resistance to anthracnose as do the stems.

The same thing is true of two lots that rated 1.3 in anthracnose resistance of stems, P. I. 168527 and P. I. 168531. Otherwise, information is lacking as to resistance of pods and seeds.

The imported lots of blue lupine listed above as highly resistant to anthracnose in the early stage of growth make fair growth but are not so vigorous as the blue lupine now grown commercially in the Southeast. They blossom about 2 weeks later and have somewhat smaller seed. Their seed are moderately hard to very hard and must be scarified before being planted. Because of low vigor and high proportion of hard seeds these imported lots will probably be unacceptable to the farmer. They are not highly resistant to the brown spot fungus (*Ceratophorum setosum* Kirchn.), but are more resistant to it than some selections of the blue lupine now grown commercially in the Southeast.

SUMMARY

Anthracnose of lupine, caused by the fungus *Glomerella cingulata*, has become more important in recent years, particularly because blue lupine (*Lupinus angustifolius*) has become the major cover-crop plant in large areas in the southeastern United States. The disease is widespread in the lupine belt, but usually is not serious except in wet seasons and in certain parts of southern Georgia, southern Alabama, and northern Florida.

The most conspicuous symptoms are blackish cankers that often girdle the stems of seedlings in the autumn and winter and brownish lesions on stems and pods in the spring. Seeds in infected pods may be badly decayed. Both the conidia and the ascospores may be present on lesions in the field when conditions are favorable for development of the fungus. Setae often are abundant but may be entirely lacking.

Infection of blue lupine is easily obtained, under suitable conditions of temperature (20°–30° C.) and high moisture, by such methods as spraying the plants with a spore or mycelial suspension or by inoculating the seed before planting or injecting spores or mycelium into the stems or cotyledons with a hypodermic needle.

Earlier research showed that *Glomerella cingulata* from lupine can attack fruits of peach and apple and cotyledons of seedlings of coffeeweed and *Crotalaria spectabilis*. Isolates of the fungus from horsechestnut, sorghum, black medic, *Arachis* sp., and kudzu proved to be weak to vigorous parasites on blue lupine.

The causal fungus is seed-borne, and can survive for several months in stored seed. Evidence was found that it is largely or entirely dead by the second planting time, or after about 18 months' storage.

Optimum temperature for mycelial growth of the strain of this fungus used on agar is near 25° C. The mycelium made no growth, but was not killed, in 13 days at 4.2°. Very slight growth was made in 5 days at 37.5°, but death occurred in that time at 39.5°. At 51° the spores were killed in 10 minutes. Conidia that were removed from pods and kept dry died in less than a month.

The fungus in the seed can be killed by exposing the seed to an air temperature of 75° C. for 3 hours. This heat treatment somewhat retards germination and may cause hardening of some seeds. Seed must be dried in advance to a moisture content of approximately 11 or 12 percent to withstand this temperature. Hot-water treatment is not a practical method of controlling the anthracnose; water hot enough to kill the fungus within the seeds also kills many of the seeds.

It seems probable that rotation has some value in the control of anthracnose, and that a 3- or 4-year rotation is to be recommended even though other hosts may carry the fungus over the summer. The possibility that the fungus can live over the summer in parts of old lupine plants or in the soil has not been exhaustively investigated. Efforts to isolate the fungus from old stems left on the surface of the soil until late summer yielded negative results.

Spraying or dusting as a means of controlling anthracnose on the seedlings or the pods was not tested. It seems logical at present to use the crop for green manure where the disease has become widespread rather than to save it for seed.

Evidence was obtained that considerable resistance to the anthracnose fungus exists in the stems of some imported lots of lupine.

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